UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/733,306	12/08/2000	Margaret A. Schwarz	9022.20	3192

20792 7590 08/01/2008
MYERS BIGEL SIBLEY & SAJOVEC
PO BOX 37428
RALEIGH, NC 27627

EXAMINER	
HUYNH, PHUONG N	
ART UNIT	PAPER NUMBER
1644	

MAIL DATE	DELIVERY MODE
08/01/2008	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/733,306	SCHWARZ, MARGARET A.	
	Examiner	Art Unit	
	PHUONG HUYNH	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *three* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *09 May 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,2,7-11,17-19,47-54 and 58-67* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-2, 7-11, 17-19, 47-54 and 58-67* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *5/9/08*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on May 9, 2008 has been entered.

2. Claims 1-2, 7-11, 17-19, 47-54 and 58-67 are pending and are being acted upon in this Office Action.

3. Claims 1, 11, and 58 are objected to because the abbreviation "EMAP II" should be "Endothelial Monocyte Activating Polypeptide II (EMAP II)" when it first appears in the claims.

4. The amendment filed May 9, 2008 is objected to under 35 U.S.C. 132(a) because it introduces new matter into the disclosure. 35 U.S.C. 132(a) states that no amendment shall introduce new matter into the disclosure of the invention. The added material which is not supported by the original disclosure is as follows: "Since the N-terminal sequence obtained from the purified EMAP II is encoded by an internal sequence of the EMAP II clone, it was predicted that mature EMAP II (e.g., SEQ ID NO:5) results from processing from a larger polypeptide (e.g., SEQ ID NO:4) (see Stem et al., U.S. Patent No. 5,641,867, incorporated by reference herein) as well as the Sequence Listing of SEQ ID NO: 4, and SEQ ID NO: 5".

Applicant is required to cancel the new matter in the reply to this Office Action.

5. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

6. Claims 1-2, 7-11, 17-19, 47-54 and 58-67 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling only for (1) a method of improving myocardial function of a subject in need of such treatment, the method comprising administering an antibody that binds to an epitope of Endothelial Monocyte Activating Polypeptide II (EMAP II) wherein the epitope consisting of the amino acid sequence of SEQ ID NO: 1, in an amount sufficient to inhibit the anti-angiogenic activity of EMAP II in the subject, thereby improving myocardial function in the subject, and (2) the method mentioned above wherein the subject is afflicted with myocardial ischemia, **does not** reasonably provide enablement for a method of *facilitating vascular growth in cardiac muscle* of a *human subject* in need of such treatment, comprising: inhibiting any and all *activity* of EMAP II of SEQ ID NO:4 or SEQ ID NO: 5 in said human subject by administering any antibody such as polyclonal antibody that specifically binds to EMAP II of SEQ ID NO:4 or SEQ ID NO: 5 as set forth in claims 1-2, 7-10, 47, 49, 51, 53, and 58-64, and (2) a method of facilitating *vascular growth in cardiac muscle tissue* of a human subject in need of such treatment, said method comprising: administering to said human subject any antibody such as any polyclonal antibody that specifically binds to EMAP II of SEQ ID NO:4 or SEQ ID NO: 6 in an amount effective to *promote blood vessel formation in said cardiac muscle* as set forth in claims 11-19, 48, 50, 52, 54, and 65-67. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in **scope** with these claims.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention. The specification disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without an undue amount of experimentation.

Claims 1-2, 7-10, 47, 49, 51, 53, and 58-64 encompass a method of *facilitating vascular growth in cardiac muscle* of a *human subject* in need of such treatment, comprising: inhibiting any and all activity of EMAP II of SEQ ID NO:4 or SEQ ID NO: 5 in said human subject by administering an antibody that specifically binds to EMAP II of SEQ ID NO:4 or SEQ ID NO: 5.

Claims 11-19, 48, 50, 52, 54, and 65-67 encompass a method of facilitating *vascular growth in cardiac muscle tissue* of *a human subject* in need of such treatment, said method comprising: administering to said human subject an antibody that specifically binds to EMAP II of SEQ ID NO:4 or SEQ ID NO: 6 in an amount effective to *promote blood vessel formation in said cardiac muscle.*

Enablement is not commensurate in scope with methods of stimulating vascular growth in cardiac muscle of a human subject by inhibiting any activity of EMAP II or promoting blood vessel formation in cardiac muscle of a human subject by administering any antibody that binds to EMAP II of SEQ ID NO: 4, 5 or 6.

The specification discloses only administering polyclonal that binds to EMAP II epitope consisting of the amino acid sequence of SEQ ID NO: 1 following myocardial infarction generated in rat by ligation of the left anterior descending artery (LAD). Rats were administered polyclonal antibody that binds to an epitope of SEQ ID NO: 1 on EMAP II one hour after post-infraction and every third day for a total of three doses. The treatment improved cardiac output that was due to an improvement in stroke volume in those rats receiving EMAP II antibody and it reaches statistical significance only at 28 days postoperatively. The disclosure postulates that inhibition of EMAP II's anti-angiogenic effect improves diastolic function and ventricular contractility. At the time of filing, the specification discloses only antibodies such as monoclonal and polyclonal antibodies generated from the peptide consisting of the amino acid sequence DAFPGEPDKELNP of SEQ ID NO: 1 wherein the antibody binds specifically to endothelial-monocyte activating polypeptide II. The specification as filed in Example I, see pages 11-12, Applicant demonstrated an improvement in myocardial function in rats comprising the administration of a rabbit EMAP II antibody, *post operatively.*

However, at the time of filing, there is no disclosure of administering any human subject afflicted with atherosclerosis, any myocardial disease, cardiomyopathy or cardiac hypertrophy any antibody, any antibody such as polyclonal antibody that binds to EMAP II of SEQ ID NO: 4, 5, or 6 mentioned above resulted in stimulating vascular growth in cardiac muscle or promote blood vessel formation in cardiac muscle of any human subject. There is no showing of any antibody that binds to EMAP II resulted in inhibiting any and all activity of EMAP II, much less facilitating vascular growth in cardiac muscle in vitro or in vivo such as in a human subject. Applicant was not in possession of the full scope of antibody which functions to inhibit any and all activity of EMAP II that is required for the practice of the claimed methods. Further, there is

no evidence to suggest that administering any antibody that binds to EMAP II of SEQ ID NO: 4 or SEQ ID NO: 6 resulted in promoting blood vessel formation in the cardiac muscle *in vitro* or *in vivo* in subject such as a human subject.

First, the use of rat model of myocardial infraction is not an appropriate model for any and all myocardial diseases, atherosclerosis, cardiaomyopathy or cardiac hypertrophy.

Thompson et al (J Surgical Research 116: 156-164, 2004; PTO 1449) teach ligation of the left anterior descending artery (LAD) is a rat model of myocardial infarction afflicted with ischemia; it is not a model for atherosclerosis, any myocardial disease or any cardiac hypertrophy (see page 157, col. 1, in particular). Further, following myocardial infraction (ischemia), EMAP II was localized to predominantly to the inflammatory infiltration of inflammatory cells of the infarct region and fibroblast, not cardiac muscle (see page 159, col. 2, page 160, col. 1, in particular). There is insufficient guidance the specific role of EMAP II in the disease cardiac muscle, especially in adult, let alone facilitating vascular growth in cardiac muscle or promote blood vessel formation in the specific tissue such as cardiac muscle. Further, there is no evidence that human EMAP II having the sequence of SEQ ID NO: 4, 5 or 6 is expressed in human adult diseased cardiac tissue such that inhibition of EMAP II function via antibody administration would effectively facilitate vessel formation in the cardiac tissue such that amelioration of the diseased state in the human would be achieved.

Second, the disclosure of the *in vivo* use of polyclonal antibody that binds to a peptide consisting of the amino acid sequence of SEQ ID NO: 1 in rat is not sufficient to provide predictive guidance for the use of any antibodies targeting that binds to EMAP II of SEQ ID NO: 4, or 5 *in vivo*. This is because the knowledge of the *in vivo* processing of EMAP II in cardiac muscle is unclear; the art is unclear as to what form of EMAP II would be present in diseased adult cardiac muscle such that administration of an antibody targeting the biologically active form of EMAP II would effectively block its function such that vessel formation could be facilitated.

It has been well known to those skilled in the art at the time the invention was made that minor structural differences among structurally related compounds or compositions can result in substantially different biological or pharmacological activities. It is known in the art that binding specificity of antibodies differ respect to whether the immunogen is a peptide or a full-length sequence.

Kuby et al (Immunology, Second edition, pages 86-96, 1994; PTO 892) teach that antibody epitopes (B cell epitopes) are not linear and are comprised of complex three-

dimensional array of scattered residues which will fold into specific conformation that contribute to binding (See Kuby 1994, page 94, in particular). Immunization with a peptide fragment derived from a full-length polypeptide may result in antibody specificity that differs from the antibody specificity directed against the native full-length polypeptide.

Further, given the polyclonal nature of polyclonal antibody, it is unpredictable which undisclosed antibody is efficacious for the claimed method.

Given the many function of EMAP-II ranging from apoptosis, chemoattractant for leukocytes and macrophages and induction of TNF-like thrombohemorrhage, the lack of clear guidance given the in the prior art for the use of antibodies that bind to EMAP II to facilitate the growth of cardiac muscle and promote blood vessel formation in cardiac muscle in human subject suffering from a variety of myocardial disease, the limited working example, the breadth of the claims, which encompass innumerable possible antibodies, and the amount of experimentation required to determine each possible species individually, it would require undue experimentation to use the invention in a manner commensurate in scope with the claims. See page 1338, footnote 7 of Ex parte Aggarwal, 23 USPQ2d 1334 (PTO Bd. Pat App. & Inter. 1992).

In re wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988), the decision of the court indicates that the more unpredictable the area is, the more specific enablement is necessary. In view of the quantity of experimentation necessary, the limited working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims, it would take an undue amount of experimentation for one skilled in the art to practice the claimed invention.

7. Claims 1-2, 7-11, 17-19, 47-54 and 58-67 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. **This is new matter**.

The recitation of "human subject" in claims 1, 11, 58, and 65 has no support in the specification and the claims as originally filed.

Further, the recitation of "…antibody that specifically binds to SEQ ID NO: 4" in claims 1, and 11, the recitation of "…antibody that specifically binds to EMAP II of SEQ ID NO: 5" in claim 58 and the recitation of "…antibody that specifically binds to EMAP II of SEQ ID NO: 6" in newly added claim 65 has no support in the specification and the claims as originally filed.

The specification as originally filed discloses only one peptide consisting of the amino acid sequence of SEQ ID NO: 1 and antibody made using the peptide of SEQ ID NO: 1 for a method of improving myocardial function of a subject. The specification at page 4 discloses "Examples of EMAP II antibodies are provided in U.S. Patent No. 5,641,867 to Stern et al., the disclosure of which is incorporated herein by reference". Although the specification discloses other antibodies such as monoclonal antibody by incorporating the antibody that bind to EMAP II from US Pat 5,641,867, it is noted that the '867 patent does not disclose any antibody that binds to EMAP II of SEQ ID NO: 4, SEQ ID NO: 5, or SEQ ID NO: 6 as now claimed. The '867 patent discloses polyclonal antibodies that bind to endothelial monocyte activating polypeptide II with an apparent molecular weight of about 20 kilodaltons by SDS-PAGE; and the antibody is made by immunizing a rabbit with a peptide consisting of the amino acid sequence of SEQ ID NO: 1 or 2 coupled to keyhole limpet hemocyanin (see claims of the '867 patent).

8. Claims 1-2, 7-11, 17-19, 47-54 and 58-67 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Claims 1-2, 7-10, 47, 49, 51, 53, and 58-64 encompass a method of *facilitating vascular growth in cardiac muscle* of a human subject in need of such treatment, comprising: inhibiting any and all activity of EMAP II of SEQ ID NO:4 or SEQ ID NO: 5 in said human subject by administering an antibody that specifically binds to EMAP II of SEQ ID NO:4 or SEQ ID NO: 5.

Claims 11-19, 48, 50, 52, 54, and 65-67 encompass a method of facilitating *vascular growth in cardiac muscle tissue* of a human subject in need of such treatment, said method comprising: administering to said human subject an antibody that specifically binds to EMAP II of SEQ ID NO:4 or SEQ ID NO: 6 in an amount effective to *promote blood vessel formation in said cardiac muscle.*

For claims drawn to a genus, MPEP § 2163 states the written description requirement for a claimed genus may be satisfied through sufficient description of a representative number of species by actual reduction to practice, reduction to drawings, or by disclosure of relevant, identifying characteristics, i.e., complete or partial structure or other physical and/or chemical properties, by functional characteristics coupled with a known or disclosed correlation between

function and structure, or by a combination of such identifying characteristics, method of making the claimed invention, level of skill and knowledge in the art and predictability in the art sufficient to show that applicant was in possession of the claimed genus. See *Eli Lilly*, 119 F.3d at 1568, 43 USPQ2d at 1406.

Vas-Cath Inc. v. Mahurkar, 19 USPQ2d 1111, makes clear that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention. The invention is, for purposes of the written description inquiry, whatever is now claimed." (See page 1117.) The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See Vas-Cath at page 1116.). One cannot describe what one has not conceived. See *Fiddes v. Baird*, 30 USPQ2d 1481, 1483. In *Fiddes v. Baird*, claims directed to mammalian FGF's were found unpatentable due to lack of written description for the broad class. The specification provides only the bovine sequence.

In this case, the specification discloses only administering polyclonal that binds to EMAP II epitope consisting of the amino acid sequence of SEQ ID NO: 1 following myocardial infarction generated in rat by ligation of the left anterior descending artery (LAD). Rats were administered polyclonal antibody to EMAP II one hour after post-infraction and every third day for a total of three doses. The treatment improved cardiac output that was due to an improvement in stroke volume in those rats receiving EMAP II antibody and it reaches statistical significance only at 28 days postoperatively. The disclosure postulates that inhibition of EMAP II's anti-angiogenic effect improves diastolic function and ventricular contractility. At the time of filing, the specification discloses only antibodies such as monoclonal and polyclonal antibodies generated from the peptide consisting of the amino acid sequence DAFPGEPDKELNP of SEQ ID NO: 1 wherein the antibody binds specifically to endothelial-monocyte activating polypeptide II. The specification as filed in Example I, see pages 11-12, Applicant demonstrated an improvement in myocardial function in rats comprising the administration of a rabbit EMAP II antibody, *post operatively.*

At the time of filing, Applicant is not in possession of any antibody, any antibody such as polyclonal antibody that binds to EMAP II of SEQ ID NO: 4, 5, or 6 that inhibits any and all *activity* of EMAP II of SEQ ID NO: 4 in a human subject for a method of facilitating vascular growth in cardiac muscle or promote blood vessel formation in the cardiac muscle of a human subject afflicted with myocardial ischemia, atherosclerosis, any and all myocardial disease,

cardiomyophathy or cardiac hypertrophy. There is no disclosure of administering any human subject afflicted with atherosclerosis, any myocardial disease, cardiomyopathy or cardiac hypertrophy any antibody, any antibody such as polyclonal antibody that binds to EMAP II of SEQ ID NO: 4, 5, or 6 mentioned above that resulted in stimulating vascular growth in cardiac muscle or promoting blood vessel formation in cardiac muscle. There is no showing of any antibody that binds to EMAP II resulted in inhibiting any and all activity of EMAP II, much less facilitating vascular growth in vitro or in vivo such as in a human subject.

It is known in the art at the time the invention was made that antibody made with a peptide may resulted in different binding specificity than antibody made with a full-length polypeptide.

Kuby et al (Immunology, Second edition, pages 86-96, 1994; PTO 892) teach that antibody epitopes (B cell epitopes) are not linear and are comprised of complex three-dimensional array of scattered residues which will fold into specific conformation that contribute to binding (See Kuby 1994, page 94, in particular). Immunization with a peptide fragment derived from a full-length polypeptide may result in antibody specificity that differs from the antibody specificity directed against the native full-length polypeptide.

At the time of filing, Applicant was not in possession of the full scope of antibody which functions to inhibit any and all activity of EMAP II that is required for the practice of the claimed methods. Further, there is no evidence to suggest that administering any antibody that binds to EMAP II of SEQ ID NO: 4 or SEQ ID NO: 6 resulted in promoting blood vessel formation in the specific tissue such as cardiac muscle *in vitro* or *in vivo* in subject such as a human subject.

With the exception of the specific antibody that binds specifically to a peptide DAFPGEPDKELNP consisting of the amino acid sequence of SEQ ID NO: 6 immunized with a peptide CDAFPGEPDKELNP consisting of the amino acid sequence of SEQ ID NO: 1 for improving myocardial function of a subject in need of such treatment or purifying recombinant EMAP II, in the specification as filed, there is insufficient written description about the binding specificity of any and all antibodies that bind to EMAP II of SEQ ID NO: 4, or SEQ ID NO:5 for the facilitation of vascular growth in cardiac muscle or promote blood vessel formation in the cardiac muscle of a human subject afflicted with myocardial ischemia, atherosclerosis, myocardial disease, cardiomyopathy or cardiac hypertrophy using any antibody that binds to SEQ ID NO: 4 or 5. Even if the antibody binds to the peptide of SEQ ID NO: 1 or SEQ ID NO: 6, there is no showing of any antibody that binds to EMAP II of SEQ ID NO: 1 or SEQ ID NO: 6

resulted in inhibiting any and all activity of EMAP II of SEQ ID NO: 4 or SEQ ID NO: 5, let alone facilitating the vascular growth *in vitro* or *in vivo* such as in a human subject.

The specification as filed discloses only a composition comprising an antibody that binds only human EMAP II peptide consisting of the amino acid sequence CDAFPGEPDKELNP (SEQ ID NO: 1) or DAFPGEPDKELNP (SEQ ID NO: 6), one of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species of antibody to describe the genus of antibody, let alone inhibiting any and all "activity" of EMAP II of SEQ ID NO: 4 or 5 or 6, thereby facilitating vascular growth in cardiac muscle or promote blood vessel formation in the cardiac muscle of subject for the treatment of myocardial ischemia, atherosclerosis, myocardial disease, cardiomyopathy or cardiac hypertrophy using *any* antibody that binds to SEQ ID NO: 4, SEQ ID NO: 5 or SEQ ID NO: 6.

See MPEP § 2163, which states "[A] biomolecule sequence described only by a functional characteristic, without any known or disclosed correlation between that function and the structure of the sequence, normally is not a sufficient identifying characteristic for written description purposes, even when accompanied by a method of obtaining the claimed sequence."

See also January 5, 2001 (Vol. 66, No. 4, pages 1099-1111) Federal Register for the Guidelines for Examination of Patent Applications Under the 35 USC 112 ¶ 1, "Written Description" Requirement. These guidelines state: "[T]o satisfy the written description requirement, a patent specification must describe the claimed invention in sufficient detail that one skilled in the art can reasonably conclude that the inventor had possession of the claimed invention. An applicant shows possession of the claimed invention by describing the claimed invention with all of its limitations using such descriptive means as words, structures, figures, diagrams, and formulas that fully set forth the claimed invention. Possession may be shown in a variety of ways including description of an actual reduction to practice, or by showing that the. invention was "ready for patenting" such as by the disclosure of drawings or structural chemical formulas that show that the invention was complete, or by describing distinguishing identifying characteristics sufficient to show that applicant was in possession of the claimed invention."

In the instant case it is clear that Applicant was not in possession of the full scope of possible polyclonal or monoclonal antibodies, humanized or chimeric antibodies isolated

form any suitable source, such as chicken, goat, rabbit, horse, etc., wherein said antibody specifically binds EMAP II of SEQ ID NO: 4, SEQ ID NO: 5 or SEQ ID NO: 6 for the claimed methods.

9. No claim is allowed.

10. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Phuong Huynh, Ph.D. whose telephone number is (571) 272-0846. The examiner can normally be reached Monday through Thursday from 9:00 a.m. to 6:30 p.m. and alternate Friday from 9: 00 a.m. to 5:30 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Eileen B O'Hara can be reached on (571) 272-0878. The IFW official Fax number is (571) 273-8300.

11. Any information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/Phuong Huynh/
Primary Examiner, Art Unit 1644
July 18, 2008